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L1	6876	((544/54,97,121,316,360,366,383) or (514/227.2,228.8,274,255.02,254.02, 235.8,253.1)).CCLS.	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	OR	OFF	2005/10/03 13:11

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#4	Search **detrusor hyperactivity**	12:38:20	152
#3	Search **#1 and #2**	12:34:02	0
#2	Search **unstable bladder**	12:33:52	524
#1	Search **(m2 or m3) and (muscarinic or muscurine)**	12:32:56	3891

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1: Scand J Urol Nephrol Suppl. 2000;(206):1-44.

Related Articles, Links

Enuresis--background and treatment.

Neveus T, Lackgren G, Tuvemo T, Hetta J, Hjalmas K, Stenberg A.

Dept of Women's and Children's Health, Uppsala University Children's Hospital, Sweden.

Nocturnal urinary continence is dependent on 3 factors: 1) nocturnal urine production, 2) nocturnal bladder function and 3) sleep and arousal mechanisms. Any child will suffer from nocturnal enuresis if more urine is produced than can be contained in the bladder or if the detrusor is hyperactive, provided that he or she is not awakened by the imminent bladder contraction. Urine production is regulated by fluid intake and several interrelated renal, hormonal and neural factors, foremost of which are vasopressin, renin, angiotensin and the sympathetic nervous system. Detrusor function is governed by the autonomic nervous system which under ideal conditions is under central nervous control. Arousal from sleep is dependent on the reticular activating system, a diffuse neural network that translates sensory input into arousal stimuli via brain stem noradrenergic neurons. Disturbances in nocturnal urine production, bladder function and arousal mechanisms have all been firmly implicated as pathogenetic factors in nocturnal enuresis. The group of enuretic children are, however, pathogenetically heterogeneous, and two main types can be discerned: 1) Diuresis-dependent enuresis - these children void because of excessive nocturnal urine production and impaired arousal mechanisms. 2) Detrusor-dependent enuresis - these children void because of nocturnal detrusor hyperactivity and impaired arousal mechanisms. The main clinical difference between the two groups is that desmopressin is usually effective in the former but not in the latter. There are two first-line therapies in nocturnal enuresis: the enuresis alarm and desmopressin medication. Promising second-line treatments include anticholinergic drugs, urotherapy and treatment of occult constipation.

Publication Types:
- Review

PMID: 11196246 [PubMed - indexed for MEDLINE]

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1: Life Sci. 1999;64(6-7):419-28. Related Articles, Links

Muscarinic receptor subtypes modulating smooth muscle contractility in the urinary bladder.

Hegde SS, Eglen RM.

Department of Urogenital Pharmacology, Center for Biological Research, Roche Bioscience, Palo Alto, CA 94304, USA.

Normal physiological voiding as well as generation of abnormal bladder contractions in diseased states is critically dependent on acetylcholine-induced stimulation of contractile muscarinic receptors on the smooth muscle (detrusor) of the urinary bladder. Muscarinic receptor antagonists are efficacious in treating the symptoms of bladder hyperactivity, such as urge incontinence, although the usefulness of available drugs is limited by undesirable side-effects. Detrusor smooth muscle is endowed principally with M2 and M3 muscarinic receptors with the former predominating in number. M3 muscarinic receptors, coupled to stimulation of phosphoinositide turnover, mediate the direct contractile effects of acetylcholine in the detrusor. Emerging evidence suggests that M2 muscarinic receptors, via inhibition of adenylyl cyclase, cause smooth muscle contraction indirectly by inhibiting sympathetically (beta-adrenoceptor)-mediated relaxation. In certain diseased states, M2 receptors may also contribute to direct smooth muscle contraction. Other contractile mechanisms involving M2 muscarinic receptors, such as activation of a non-specific cationic channel and inactivation of potassium channels, may also be operative in the bladder and requires further investigation. From a therapeutic standpoint, combined blockade of M2 and M3 muscarinic receptors would seem to be ideal since this approach would evoke complete inhibition of cholinergically-evoked smooth muscle contractions. However, if either the M2 or M3 receptor assumes a greater pathophysiological role in disease states, then selective antagonism of only one of the two receptors may be the more rational approach. The ultimate therapeutic strategy is also influenced by the extent to which pre-junctional M1 facilitatory and M2 inhibitory muscarinic receptors regulate acetylcholine release and also which subtypes mediate the undesirable effects of muscarinic receptor blockade such as dry mouth. Finally, the consequence of muscarinic receptor blockade in the central nervous system on the micturition reflex, an issue which is poorly studied and seldom taken into consideration, should not be ignored.

Publication Types:
- Review

- Review, Tutorial

PMID: 10069505 [PubMed - indexed for MEDLINE]
